

DEPARTMENT OF HEALTH & HUMAN SERVICES

84 JAN 33 AM 10: 39

HEADQUARTERS

Public Health Service
National Institute for Occupational
Safety and Health
Robert A. Taft Laboratories
4676 Columbia Parkway
Cincinnati OH 45226-1998

January 26, 2004 HETA 2004-0024

Gary Morris Chief, Seattle Fire Department 301 Second Ave South Seattle, Washington 98104-2680

Mr. Paul Atwater
President, IAFF Local 27
517 2nd Ave West
Seattle, Washington 98119-3927

Dear Chief Morris and Mr. Atwater:

The National Institute for Occupational Safety and Health (NIOSH) received your independent requests for a Health Hazard Evaluation (HHE) in October 2003. Your requests focused on the health concerns of fire fighters operating out of Station 31 of the Seattle Fire Department (SFD). The health conditions of primary concern were cancer and alopecia (hair loss). On December 18, 2003, NIOSH conducted a site-visit at Station 31, interviewed Station 31 fire fighters, and met with various officials of the Fire Department, the County Health Department, the Mayor's office, and the Local Union. I applaud the efforts of both management and union to address these health concerns in the spirit of cooperation and transparency. After providing some brief background information regarding cancer and alopecia, this letter summarizes my recommendations to your task force and various panels addressing exposures and health issues of fire fighters assigned to Station 31.

Background

Cancer

It is useful, when discussing cancer and cancer clusters, to review certain facts. Cancer is common in the United States: one in two men, and one in three women will develop some type of cancer in their lifetime. One of every four deaths in the United States is from cancer. Many factors play a role in the development of cancer. Some of the factors include: (a) personal characteristics such as age, sex, and race, (b) family history of cancer, (c) diet, (d) personal habits such as cigarette smoking and alcohol consumption, (e) the presence of certain medical conditions, (f) exposure to cancer-causing agents in the environment, and (g) exposure to cancer-causing agents in the workplace. Finally, although some causes of some types of cancer are known, a lot remains unknown.

Cancer Clusters

Cancers often appear to occur in clusters, which scientists define as an unusual concentration of cancer cases in a defined area or time. A cluster also occurs when the cancers are found among workers of a different age or sex group than is usual. The cases of cancer may have a common cause or may be the coincidental occurrence of unrelated causes. The number of cases may seem high, particularly among the small group of people who have something in common with the cases, such as working in the same building. Although the occurrence of a disease may be random, diseases often are not distributed randomly in the population, and clusters of disease may arise by chance alone. iii

Studies of Cancer among Firefighters

Fire fighting exposes fire fighters to a number of hazardous substances including many potential human carcinogens. These exposures occur not only during "knockdown," but also during the overhaul phase of fire suppression when fire fighters are less likely to wear their self-contained-breathing-apparatus (SCBA). Most, although not all, incidence and mortality studies have found that fire fighters have increased rates for some specific types of cancer. Accordingly, by 2001, twenty States had adopted some type of presumptive disability statutes with regard to cancer. One of these studies, in fact, was conducted among Seattle fire fighters. This study found Seattle fire fighters had excess cancer rates for stomach, pancreas, biliary passages & liver, prostate, skin, and lymphatic & haematopoietic systems. However, none of these excesses were statistically significant, and therefore could have arisen by chance. Finally, the incidence and mortality studies cited above do not link the cancer to a specific carcinogen and, in general, assume hazardous exposure occurred during fire suppression, not at the fire station.

Alopecia

Alopecia is the loss of hair. There are many reasons for hair loss, which are classified into scarring (cicatricial) and non-scarring (noncicatricial) types (see Appendix A for a more detailed description). While a number of chemicals in the industrial work environment have been associated with some types of alopecia (see Appendix A), the environmental evaluation (described below) suggests these chemicals are not present in Station 31. For fire fighters with alopecia, I recommend an evaluation by their primary care physicians, and no further environmental testing. The remainder of this letter addresses the cancer cluster concerns.

Expert Panels

Industrial Hygiene

In the fall of 2003, the SFD contracted with Prezant Associates to conduct an environmental assessment of Station 31. Mr. David Chawes, a Certified Industrial Hygienist, developed the sampling strategy and protocol. Results of the testing are available on the SFD Station 31 Website [www.cityofseattle.net/fire/station31] and were presented to Station 31 members during the NIOSH site-visit on December 18, 2003. Environmental testing was very extensive. The results revealed some unexpected findings unrelated to the health issues of concern; for example,

Page 3 - Chief Morris & Mr. Atwater

the presence of lead in the basement soil. However, for all the carcinogens tested (arsenic, asbestos, benzene, cadmium, hexavalent chromium, diesel particulate, polycyclic aromatic hydrocarbons, radon gas, and crystalline silica), none were at levels considered hazardous (i.e. no levels approached their "action limits"). Actions limits are the permissible exposure limits (PEL), recommended exposure limits (REL), threshold limit values (TLV), or recommendations set by WISHA, NIOSH, ACGIH, and EPA, respectively.

Interpretation of the environmental findings has some limitations. Environmental tests conducted in 2003 cannot rule out hazardous exposures during previous years. In addition, most of the "action limits" are based on an eight-hour exposure period, rather than a 24-hour shift experienced by most fire fighters. Nonetheless, with the exception of the lead contamination in the soil, there is no evidence of hazardous exposures currently in Station 31. Closing Station 31 is not justified. Based on the findings I recommend the following:

- (1) the lead-contaminated soil be removed and/or encapsulated;
- (2) with the exception of some follow-up lead testing, additional environmental sampling is not indicated;
- (3) although the tailpipe exhaust system works to limit exposure to diesel exhaust, this system could be supplemented with any, or all, of the following:
 - Install doors with weather stripping from the garage to the offices and living quarters to
 prevent infiltration of diesel fumes.
 Keep the living quarters under positive pressure to prevent entry of exhaust into the living
 quarters.
 - Always open the garage doors before vehicles are started.
 - Keep fire engine operation inside the garage to an absolute minimum.
 - Keep doors between the garage and other areas of the firehouse closed.

 Perform regular engine maintenance to minimize diesel particulate emissions.

Epidemiology Panel

In the fall of 2003, the SFD organized an epidemiology panel to determine what type, if any, epidemiologic study should be undertaken. Members of this panel are listed on the SFD Station 31 Website [www.cityofseattle.net/fire/station31] with representatives from the public health and academic communities, as well as representatives from management and labor. At the request of the epidemiology panel, the SFD is developing a list of all fire fighters who ever worked at Station 31 which opened at the end of 1974. This list will be cross-referenced with the County's cancer registry to identify all cancer cases among fire fighters who ever worked at Station 31. This list of cancer cases could then be compared to the informal roster gathered by Station 31 members to allow an individual medical record review for discrepancies.

For the purposes of further analysis or studies, the panel will subsequently determine the minimum time period (possibly one year) to be designated as a "Station 31 Fire Fighter." The panel could then compare the observed number of cancer cases in Station 31 fire fighters with the

Page 4 - Chief Morris & Mr. Atwater

expected number of cancer cases for the County, State, or United States, after adjusting for potential confounders (e.g. age, gender, race, etc). Based on the numbers and types of cancers found, the panel will determine if this type of study, known as a Standardized Incident Ratio (SIR), is appropriate. It is important to note that during its 28-year history, relatively few fire fighters worked at Station 31. Therefore, even if more cancer cases are observed than are expected, this increase is unlikely to achieve statistical significance, making it difficult to draw conclusions.ⁱⁱⁱ

Medical Screening Panel

Again, I applaud the Seattle Fire Department for composing a panel with experience and expertise, and for encouraging union participation. The medical screening panel was scheduled to meet after results from the environmental testing were available. NIOSH defers to the panel to determine which, if any, medical screening tests are appropriate for Station 31 members. However, I do not support the use of "total body scans" or computerized tomography (CT) scans as a screening test for Station 31 members – an idea proposed by some members of Station 31. While these scans can be indicated in the diagnostic work-up of individual patients, they are not helpful or indicated as a screening test.

While the panel's purpose is to determine what, if any, medical testing should be conducted on Station 31 members, the SFD may want to take a broader perspective. For example, should all Seattle fire fighters be medically screened on a periodic basis to address fitness-for-duty issues? Three consensus standards from the National Fire Protection Association (NFPA) recommend annual medical evaluations and screening tests:

NFPA 1500, Standard on Fire Department Occupational Safety and Health Program,^x NPFA 1582, Standard on Comprehensive Occupational Medical Program for Fire Departments,^{xi} NFPA 1583, Standard on Health-Related Fitness Programs for Fire Fighters,^{xii}

The SFD and the Union should review these standards to identify applicable elements for their Department. Other large-city negotiated programs can also be reviewed as potential models. In addition to benefiting employee health, wellness programs in other industries have been shown to be cost effective, typically by reducing the number of work-related injuries and lost work days. XIII XIV XV A similar cost savings has been reported by the wellness program at the Phoenix Fire Department, where a 12-year commitment has resulted in a significant reduction in disability pension costs. XVI

I here this information is useful for you. This letter serves to close the file for this project. For the purpose of informing affected employees, copies of this report should be posted by the SFD in a prominent place accessible to the employees for a period of 30 calendar days. If you have any questions, please do not hesitate to contact me at 513-841-4386.

Page 5 - Chief Morris & Mr. Atwater

Sincerely yours,

Thomas Hales, MD, MPH
Senior Medical Epidemiologist
Hazard Evaluations and Technical
Assistance Branch
Division of Surveillance, Hazard
Evaluations and Field Studies

- Jales W

ce: Rich Duffy, IAFF
Ken Tipler, SFD
Edsonya Charles, Office of the Mayor
David Solet, Seattle and King County Health Dept

REFERENCES

- vi Austin CC, Dussault G, Ecobichon DJ [2001]. Municipal firefighter exposure groups, time spent at fires, and use of self-contained-breathing-apparatus. *Am J Ind Med* 40:683-692.
- vii Guidotti TL [1995]. Occupational mortality among firefighters: assessing the association. J Occup Environ Med 37:1348-1356.
- viii No author [2001]. Presumptive disability bill for federal firefighters introduced. Fire Chief Aug1, online copy accessed on 1/14/2004 http://firechief.com/ar/firefighting presumptive disability bill/index.htm

- ^x NFPA [1997]. NFPA 1500, Standard on fire department occupational safety and health program. Quincy, MA: National Fire Protection Association.
- xi NFPA [2003]. NFPA 1582, Standard on Comprehensive Occupational Medical Program for Fire Departments. Quincy, MA: National Fire Protection Association.
- NFPA [2000]. NFPA 1582, Standard on Standard on Health-Related Fitness Programs for Fire Fighters. Quincy, MA: National Fire Protection Association.

¹ American Cancer Society [2004]. Cancer Statistic Presentaion 2004. http://www.cancer.org/docroot/pro/content/pro 1 1 Cancer Statistics 2004 presentation.asp Accessed on 1/14/04, Slides 18 & 19.

ii American Cancer Society [2004]. Cancer Statistic Presentation 2004. http://www.cancer.org/docroot/pro/content/pro 1 1 Cancer Statistics 2004 presentation.asp Accessed on 1/14/04, Slide 2.

iii MMWR [1990]. Guidelines for Investigating Clusters of Health Events. 39 (RR-11); 1-16 http://www.cdc.gov/epo/mmwr/preview/mmwrhtml/00001797.htm
Accessed on 1/14/04

iv Lees PSJ [1995]. Combustion Products and Other Firefighter Exposures. In: Orris P, Melius J, Duffy R, Eds. Fire Fighters Safety and Health. Occupational Medicine State of the Art Reviews. Philadelphia, PA: Hanley & Belfus, Inc. 10(4): 691-706.

^v Bolstad-Johnson DM, Burgess JL, Crutchfield CD, Storment S, Gerkin R, Wilson JR [2000]. Characterization of firefighter exposures during fire overhaul. *Am Ind Hyg J* 61:636-641.

ix Rosenstock L, Demer P [1991]. Northwest Firefighters Mortality Study: 1945-1989. Federal Emergency Management Agency, United States Fire Administration. FA-105.

xiii Maniscalco P, Lane R, Welke M, Mitchell J, Husting L [1999]. Decreased rate of back injuries through a wellness program for offshore petroleum employees. J Occup Environ Med 41:813-820.

xiv Stein AD, Shakour SK, Zuidema RA [2000]. Financial incentives, participation in employer sponsored health promotion, and changes in employee health and productivity: HealthPlus health quotient program. JOEM 42:1148-1155.

xv Aldana SG [2001]. Financial impact of health promotion programs: A comprehensive review of the literature. Am J Health Promot 15:296-320.

xvi Unpublished data [1997]. City Auditor, City of Phoenix, AZ. Disability retirement program evaluation. Jan 28, 1997

Appendix A - Alopecia

Alopecias are classified into cicatricial (scarring) and noncicatricial types. Examples of causes of cicatricial alopecia, which lead to destruction of the hair follicles, include--developmental defects and hereditary disorders, infections, neoplasms, physical/chemical agents (such as radiation), and certain dermatoses. Noncicatricial alopecias can be more complicated, in a pathological and diagnostic sense, and can lead to--1) situations where hair bulbs transform from a mature stage (terminal follicle) to a premature stage (vellus) resulting in short, fine hairs, or 2) situations where many hair follicles abnormally enter the telogen (resting) phase of the hair cycle and then shed together. Examples of the types of noncicatricial alopecia include--andro-genetic alopecia; hereditary syndromes; alopecia areata; traumatic alopecia (traction); and, noncicatricial alopecia associated with systemic diseases or processes (telogen effluvium, nutritional/metabolic deficiency states, endocrine diseases, drugs and chemical agents, infections).

Andro-genetic alopecia (aga) (often referred to as common balding in men and hereditary thinning in women) is the most common cause of hair thinning in both men and women and results from a gradual progression of terminal follicles to vellus-like follicles. In females this condition usually involves the central scalp with a diffuse thinning of the hair; very rarely does it progress to a complete loss of hair in females. Hair loss may occur in waves, with periods of heavier hair loss that may last for several months. There is at present no effective treatment for aga. The diagnosis is based upon history, familial incidence (although the mode of inheritance is questionable), and the pattern of hair loss. In women, further hormonal evaluation should be considered if the affected individual has one or more of the following symptoms; menstrual irregularities, infertility, hirsutism, severe cystic acne, virilization, or galactorrhea. In some instances, to exclude other treatable causes of hair thinning, other laboratory tests may be indicated (e.g. thyroid tests, blood iron levels, blood counts, and others). Hair analysis for protein, trace elements, and minerals are not appropriate tests. Alopecia areata (aa) is one of the more common non-scarring alopecias. It can range in severity from localized patches of hair loss, to complete loss of scalp hair (alopecia totalis), to generalized loss of body hair (alopecia universalis). Males and females are equally affected, and it is estimated that about one percent of the population will have at least one episode of as by age 50.² As can occur at any age, although the peak incidence is in the third to fifth decades. Most commonly, as presents as one or more round or oval patches of total hair loss measuring up to several centimeters in diameter. The scalp, bearded area, eyebrows, or other body sites may be involved. There is often no clinically evident abnormality to the skin surface other than the hair loss. Rarely, a mild redness (erythema) may be seen in early patches. Other findings that can occur with aa include nail abnormalities. As is sometimes associated with other conditions, including atopy, cataracts, thyroid disease, vitiligo, pernicious anemia, and Addison's disease. With a small number of patches, the majority of people with as will exhibit hair regrowth within 6 to 12 months. New patches may develop while older ones are resolving, and relapses can occur.

The exact causes of an remain unknown. Factors that have been cited as possible precipitants of an (although these are not fully supported or proven) include the following: 1) emotional stress, 2) defects in immunoregulation leading to autoimmunity, 3,4,5,3) genetic influences (30 percent of

Appendix A - Alopecia Continued

cases have a family history of aa),³ and 4) additional, possibly environmental, factors which serve as triggers for disease expression in those susceptible.⁶

Although the diagnosis of aa is usually made by the history and clinical exam, this condition has a very typical biopsy finding. Microscopic studies of the affected patches of skin show an immune reaction of blood cells (T lymphocytes and macrophages) surrounding the proximal hair bulbs in a "bee swarm-like" fashion.

Currently, there is no curative treatment for aa. The modalities that have been tried somewhat successfully include topical, intralesional, or systemic corticosteroids (systemic corticosteroids are used less often because of side effects); topical irritants such as anthralin, dinitrochlorobenzene, or squaric acid dibutyl ester; and localized ultraviolet therapy together with psoralen photosensitizers (PUVA therapy).

There is no direct evidence that occupational or environmental chemical exposures can cause aa. In one report on the investigation of an outbreak of aa in an oil refinery, no cause for the aa in the 12 affected workers could be found. The authors suggest a hypothesis "that under certain conditions alopecia areata can be infectious or contagious, or that exposure to some unknown substance reduces resistance to the disease." Another report describes seven cases of aa diagnosed among workers in a paper factory. No definite etiology of the aa was found, but the authors conclude that "this dermatosis could be due to the professional activities of the workers." A report from Malaysia describes three cases of aa that were felt to be causally related to occupational exposures to microwave radiation. No definitive proof of this was established. Besides these descriptions of aa in occupational settings, "epidemics" of aa were described in the early 1900's in children's institutional establishments.

Telogen effluvium [or defluvium] (te) is a condition in which the hair loss is caused by large number of hairs that have entered the telogen (resting) stage of the hair growth cycle in a relatively synchronous fashion. It is usually a diffuse process, and the affected individual may notice increased hair loss that may or may not be obvious clinically. Te can be either an acute or chronic condition.

Although not always, hair loss in acute te can follow a stressful event (e.g. childbirth) by about 2 to 4 months. It may also be associated with discontinuation of birth control pills following prolonged use, discontinuation of high-dose systemic corticosteroid treatments, high fever, blood loss, shock, extreme dieting and severe psychiatric stress. Hair regrowth usually occurs within 6 to 12 months, but may not be complete. \(^1\)

Chronic te affects 30 to 60 year old women, and starts abruptly with or without a recognizable initiating factor. It may be differentiated from acute te by its long fluctuating course and from aga by its clinical and microscopic findings.¹²

Appendix A - Alopecia Continued

Other noncicatricial alopecias (usually te) can be associated with nutritional/metabolic deficiency states (e.g., deficiencies of iron, zinc, essential fatty acids, or caused by "crash dieting"), and endocrine diseases (such as hypothyroidism, hyperthyroidism, hypopituitarism, and hypoparathyroidism). Other non-scarring, usually diffuse, alopecias (including te and anagen effluvium) can be caused by occupational or environmental exposures. These alopecias are usually easily differentiated from aa. Agents that can cause such alopecias include a variety of pharmaceutical agents (these include some specific cytostatic agents, anticoagulants, and antithyroid, antihypertensive, antiepileptic, and antipsychotic drugs), vitamin A and its analogues, thallium (formerly used in pesticides), selenium, boric acid, chloroprene (used in synthetic rubber production), and mercurials (used in bleaching cream and antiseptics). Other occupational exposures that may cause a diffuse hair loss include chlorbutadine, dimethylamine, sodium sulfide, and calcium sulfide.

The medical literature includes one example of an occupational hair loss found in a nurse. The specific diagnosis was traction alopecia, or hair loss caused by hairs being pulled out by external forces. In this case description, the hair loss was caused by traction on the hair shafts exerted at the points of attachment of a nurse's cap.¹³

References for Appendix A

- 1. Bertolino AP, Freedberg IM [1993]. Disorders of epidermal appendages and related disorders. Chapter in Dermatology in General Medicine (Fitzpatrick TB, et al. [eds]. Chicago, McGraw Hill.
- 2. Price V [1991]. Alopecia areata: Clinical aspects. J Invest Dermatol 96:68S.
- 3. Sauder DN, et al., [1980]. Alopecia areata: An inherited autoimmune disease. In Hair, Trace Elements and Human Illness (Brown AC et al. [eds]). New York, Praeger.
- 4. Paus, R, Slominski A, Czarnetzki BM [1994]. Is alopecia areata an autoimmune-response against melanogenesis-related proteins, exposed by abnormal MHC class I expression in the anagen hair bulb? Yale J Biol Med 66:541-54.
- 5. Goldsmith LA [1991]. Summary of alopecia areata research workshop and future research directions. J Invest Dermatol 96:98S.
- 6. McDonagh AJ, Messengert AG [1994]. The aetiology and pathogenesis of alopecia areata. J Dermatol Sci 7 Suppl:S125-135.
- 7. Williams N, Rigert AL [1971]. Epidemic alopecia areata An outbreak in an industrial setting. J Occup Med 13:11.

Appendix A - Alopecia Continued

- 8. Roselino AM, Almeida AM, Hippolito MA, et al [1996]. Clinical-epidemiologic study of alopecia areata. Int J Dermatol 35(3):181-4.
- 9. Isa AR, Noor M [1991]. Non-ionizing radiation exposure causing ill-health and alopecia areata. Med J Malaya 46:235-238.
- 10. Fox CT [1913]. On a small epidemic of an areate alopecia. Brit J Derm 25:51-56.
- 11. Davis H [1914]. Epidemic alopecia areata. Brit J Derm 26:207-14.
- 12. Whiting DA [1996]. Chronic telogen effluvium: Increased scalp hair shedding in middle-aged women. J Amer Acad Derm 35:899-906.
- 13. Renna FS, Freedberg IM [1973]. Traction alopecia in nurses. Arch Derm 108:694-695.